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CASE REPORT

Acinetobacter baumannii meningitis in children: a case series and literature review

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Abstract

Introduction The incidence of *Acinetobacter baumannii* meningitis, which typically occurs after neurosurgery, has increased in recent years. Pediatric *Acinetobacter baumannii* meningitis due to the emergence of multidrug-resistant (MDR) and extensively drug-resistant (XDR) strains has important clinical significance.

Methodology We retrospectively reviewed the clinical course and outcome of nine cases of meningitis due to *Acinetobacter baumannii* in children and reviewed the relevant literature.

Results Seven patients had a history of neurosurgery, and the average time from the first surgery to cerebrospinal fluid (CSF) culture in these seven patients was 23.71 ± 17.43 days. Of all nine patients, four patients showed MDR isolates, two showed XDR isolates, and one showed pan-drug-resistant (PDR) isolates. Three patients received an intrathecal injection of amikacin. Two patients received intravenous colistin (5 mg/kg), and one received polymyxin B (2 mg/kg). The mean hospitalization duration was 39.44 days. Four patients eventually died: two with MDR *Acinetobacter*, one with PDR *Acinetobacter*, and one with susceptible *Acinetobacter*. Two of them still had positive CSF cultures at death.

Conclusion *Acinetobacter baumannii* meningitis is usually associated with neurosurgery and the placement of foreign material, and it usually has a high mortality. Intrathecal or intraventricular polymyxin administration is expected to be an effective choice for meningitis but requires further study.

Keywords *Acinetobacter baumannii* · Postoperative meningitis · Children · Polymyxin

Introduction

Acinetobacter is a Gram-negative coccus that is nonfermentative, strictly aerobic, catalase positive and oxidase negative. More than 30 genera [1] have been discovered, most of which are environmental microorganisms and have no human pathogenicity. Among them, *Acinetobacter baumannii, Acinetobacter calcoaceticus*, and *Acinetobacter rufi* are the most frequently reported strains. The term *A. calcoaceticus–A. Baumannii* (ACB) complex is also used because it is difficult to identify various *Acinetobacter* species based on phenotypic characteristics. ACB complexes include gene type 1 (*Acinetobacter calcoaceticus*), gene type 2 (*Acinetobacter baumannii*), gene type 3 and gene type 13TU [2]. Among them, *Acinetobacter baumannii* has the most clinical significance.

Acinetobacter baumannii can accumulate multiple drug resistance genes, resulting in multidrug resistance (MDR) and even pan-drug resistance to clinically used antibiotics. The resistance mechanisms often expressed in hospital *Acinetobacter* strains include β-lactamase [3], cell wall channels (porins) and efflux pump changes (cephalosporin, carbapenem, quinolone, tetracycline, chloramphenicol, and tigecycline resistance) [4]; gyrA and parC mutations (quinolone resistance) [5], aminoglycoside-modifying enzyme expression (aminoglycoside resistance); and PmrA and PmrB protein-coding gene mutations (polymyxin resistance) [6].

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The most common clinical manifestations of *Acinetobacter baumannii* are ventilator-associated pneumonia and bloodstream infection [7], and *Acinetobacter baumannii* can also colonize the skin, wounds, respiratory tract and gastrointestinal tract. *Acinetobacter baumannii* is rare in nosocomial meningitis, but it has certain importance in postoperative meningitis. The risk factors are neurosurgical operation, head trauma, cerebrospinal fluid (CSF) leakage, wound infection, and foreign body implantation [8]. *Acinetobacter baumannii* meningitis has a high mortality rate of up to 15–71% [9]. Due to its limited treatments and poor prognosis (high mortality and neurological sequela rates), the clinical challenges are increasing. Pediatric *Acinetobacter baumannii* meningitis is relatively rare, and a lack of clinical cases, nonspecific clinical manifestations, poor therapeutic effects, poor prognosis, and its diagnosis and treatment present great challenges to pediatricians. In this paper, we retrospectively analyzed the clinical features, drug resistance spectrum and treatment experience of 9 children with *Acinetobacter baumannii* meningitis in this children's hospital and review the relevant literature to improve the understanding of such diseases.

Materials and methods

Inclusion and exclusion criteria

A retrospective review of all *Acinetobacter baumannii* ($n=3409$) cases from the microbiology laboratory within a single regional teaching hospital, which has 1900 beds and a well-equipped pediatric intensive care unit (PICU), surgical intensive care unit (SICU), neonatal intensive care unit (NICU) and cardiac intensive care unit (CICU), between 1 July 2006 and 1 June 2018 was undertaken.

The inclusion criteria were as follows: (1) isolation of *Acinetobacter baumannii* from CSF; (2) increased white blood cells ($\geq 10\times10^6$/L) and protein (≥ 450 mg/L) and decreased glucose (≤ 2.78 mmol/L) in the CSF; and (3) clinical evidence of central nervous system (CNS) infection, fever, headache, vomiting, confusion, irritability or meningeal irritation.

The exclusion criteria were as follows: (1) non-CSF specimen and (2) pseudomeningitis, in which the CSF is culture positive in the absence of clinical and laboratory features of meningitis.

Subjects

We retrospectively analyzed the following data: age, sex, underlying condition, invasive procedure, days from first neurosurgery to diagnosis, CSF culture, antibiotic resistance category, length of stay, duration of intravenous and intrathecal antibiotic treatment, time to CSF sterilization, and outcome. If the same child had multiple positive CSF cultures, we analyzed the data for the first instance.

Strain and drug sensitivity test

We used the VITEK2 compact (an automated microbial identification system) for strain identification and drug susceptibility testing. The minimum inhibitory concentration (MIC) was measured according to the AST-GN16 susceptibility card.

Antimicrobial susceptibilities

According to 2011 European and American antibiotic resistance consensus [10], *Acinetobacter baumannii* susceptibility test results can be divided into four categories, which are described below. Common antibacterial agents for *Acinetobacter baumannii* include broad-spectrum cephalosporins (ceftazidime, cefepime), anti-*Pseudomonas* carbapenems (imipenem, meropenem), enzyme inhibitors (ampicillin sulbactam, piperacillin tazobactam, cefoperazone sulbactam), fluoroquinolones (ciprofloxacin, levofloxacin), aminoglycosides (gentamicin, tobramycin, amikacin), polymyxin (colistin, polymyxin B), and tigecycline. The four categories are as follows: MDR, resistant to at least three types of antimicrobial agents; extensively drug-resistant (XDR), resistant to most of the above drugs and sensitive to only one or two antibiotics; pan-drug-resistant (PDR), resistant to all of the above antibacterial drugs; and susceptible *Acinetobacter* (SA), sensitive to most of the above drugs.

Results

Basic characteristics (Table 1)

Among the 3409 specimens, 21 positive CSF samples were observed in 9 children: 6 males and 3 females. The male:female ratio was 2:1, and the average patient age was 52.70 ± 48.14 months. Seven patients (77.8%) had a history of neurosurgery, and the average time from the first surgery to CSF culture in 7 patients was 23.71 ± 17.43 days. The *Acinetobacter baumannii* drug susceptibility test showed that there were four MDR cases, two XDR cases, one PDR case, and two SA cases. The average hospital stay was 39.44 days. Four patients eventually died: two with MDR *Acinetobacter*, one with PDR *Acinetobacter* and one with an SA isolate.

Clinical manifestation and laboratory examination

Nine patients had clinical manifestations of fever, vomiting, and disturbance of consciousness, and five patients

Table 1 Clinical characteristics and outcome of nine cases of *Acinetobacter* meningitis

Case	Age (months)/sex	Underlying condition	Invasive procedure	Days from first neurosurgery to diagnosis	C-reactive protein (mg/L)	White cell count in CSF (*10^6/L)		Sugar content in CSF (mmol/L)	Protein content in CSF (mg/L)		CSF culture	Antibiotic resistance category	Length of stay (days)	Outcome
1	34/M	Traffic accident	Craniotomy/EVD/Ommaya reservoirs	28	16.2	430	0.5		3040.7	*A. Baumannii–A. calcoaceticus*		MDR	63	Died
2	90/M	Intracranial teratoma	Craniotomy/EVD/Ommaya reservoirs	13	16.1	20	0.43		907.9	*A. baumannii*		MDR	40	Died
3	133/F	Intracranial hemorrhage	Evacuation of hematoma/EVD	23	15.5	72	0.56		2728.8	*A. baumannii*		XDR	33	Survived
4	Neonate/M	Neonatal septicemia	NO	NO	110	18	0.23		979.3	*A. Baumannii–A. calcoaceticus*		SA	19	Survived
5	Neonate/M	Neonatal septicemia	NO	NO	100	14	0.42		754.9	*A. baumannii*		MDR	15	Survived
6	11/M	Subdural effusion	EVD	8	16.5	204	0.94		2489.5	*A. baumannii*		MDR	39	Survived
7	14/M	Traffic accident	Craniotomy	4	16.3	2780	0.06		2641.7	*A. baumannii*		XDR	36	Survived
8	87/F	Traffic accident	Craniotomy/EVD/Ommaya reservoirs	30	31	800	0.24		7499	*A. baumannii*		PDR	51	Died
9	105/F	Cerebral trauma	Craniotomy/EVD/Ommaya reservoirs	60	105	10	0.14		5985	*A. baumannii*		SA	59	Died

EVD external ventricular drain, *MDR* multidrug-resistant, *XDR* extensively drug-resistant, *PDR* pan-drug-resistant, *SA* susceptible *Acinetobacter*

had convulsions. The CSF showed an increased white cell count, with an average of 483.11×10^6/L (10-2780×10^6/L), with neutrophils as the main contributor, and decreased sugar, with an average content of 0.34 mmol/L (0.05–0.94 mmol/L). The chloride content decreased, and the protein content increased, averaging 3002.98 mg/L (754.9–7499 mg/L). Peripheral blood routinely showed an increased white blood cell count, mostly consisting of neutrophils, and an increased C-reactive protein (CRP) level, averaging 47.4 mg/L (15.5–110 mg/L).

Mixed other infections

Corynebacterium and *Penzantella* were found in the CSF of one patient. In three cases, *Acinetobacter baumannii* was also found in sputum samples.

Antimicrobial susceptibilities

The resistance of *Acinetobacter baumannii* to clinical first-line antibiotics and antibiotic selection in nine cases is shown in Table 2. In one case each, *Acinetobacter baumannii* was only sensitive to amikacin and ceftazidime, polymyxin and amikacin, polymyxin and tigecycline, amikacin and tobramycin, amikacin, and tigecycline. In addition, *Acinetobacter baumannii* was only moderately sensitive to tigecycline in one case.

Treatment and prognosis

In addition to the two cases of neonatal septicemia, the other seven cases who had terrible underlying conditions received antibiotics to prevent perioperative infection or underwent anti-infective treatment before the diagnosis of meningitis. Among these cases, four cases received meropenem combined with vancomycin, two cases received ceftriaxone, and one case received piperacillin tazobactam. When the CSF culture was positive, the treatment plan was actively adjusted according to the drug sensitivity test. Although case 5, with a diagnosis of neonatal sepsis, was only sensitive to aminoglycosides, antibiotics were not changed due to the large side effects of the drug and its good clinical treatment effect. The antibiotics were adjusted for the remaining eight patients. See Table 2 for details. Although the susceptibility test for polymyxin was not routinely performed, our clinical pediatricians tried polymyxin in three patients based on the latest clinical experience and achieved good clinical results. Two patients received intravenous colistin (5 mg/kg) and

Table 2 The antimicrobial susceptibilities and treatment of nine cases

Case	Antibiotic resistance category	Antibiotic sensitivity	MIC	Treatment (intravenous)	Treatment (intrathecal)	Days to CSF sterilization
1	MDR	Amikacin	8	Meropenem 18 days Linezolid 18 days	Amikacin 15 mg qd 18 days	15
2	MDR	Amikacin Gentamicin Polymyxin	≤2 ≤1 NO	Cefoperazone sulbactam 22 days Amikacin 28 days	Amikacin 10 mg qd, 20 days	Still positive
3	XDR	Polymyxin Tigecycline	16 1	Cefoperazone sulbactam 11 days Tigecycline 3 days Colistin 8 days	NO	8
4	SA	Cefotaxime cefoperazone sulbactam	8 ≤4	Cefotaxime 16 days Cefoperazone sulbactam 4 days	NO	4
5	MDR	Amikacin Tobramycin	≤2 ≤1	Meropenem 15 days Piperacillin tazobactam13 days	NO	4
6	MDR	Amikacin	8	Cefoperazone sulbactam 20 days Amikacin 17 days	Amikacin 10 mg qd 3 days	3
7	XDR	Tigecycline	1	Tigecycline 3 days Cefoperazone sulbactam 22 days Colistin 22 days	NO	10
8	PDR	Tigecycline Intermediate	4	Tigecycline 8 days Cefoperazone sulbactam 21 days Polymyxin 13 days	NO	22
9	SA	Cefoperazone sulbactam	27	Cefoperazone sulbactam 6 days Linezolid 6 days	NO	Still positive

MIC minimum inhibitory concentration

one received polymyxin B (2 mg/kg); all CSF culture turned negative. Three patients received an intrathecal injection of amikacin according to the drug susceptibility results. The drainage tubes of six patients were removed. Four children died, two of whom still had positive CSF cultures.

Discussion and literature review

Acinetobacter meningitis mainly occurs after neurosurgery, and the risk factors include long neurosurgical operations, CSF leakage, incision infection, foreign body implantation, external ventricle drain (EVD) placement, multiple operations, and previous antibiotic treatments [8]. A targeted literature search was performed on PubMed, the Cochrane Library database and the World of Science. Relevant publications in English published before 1 July 2018 were found through a search on PubMed using the following key search terms: "*Acinetobacter baumannii*," "meningitis," "intracranial infection," and "neurosurgery." Summarized below are 16 articles that describe 33 pediatric cases (Table 3) [11–26]. Some of the 16 articles did not provide all of the information that is described below. Among the known information, there are 13 males and 10 females, aged from newborn to 16 years, with an average age of 57.96 ± 66.32 months. All underwent neurosurgical procedures, and eleven patients underwent EVD placement. Eventually five patients (15.2%) died. These findings are consistent with our case summary results. Seven of our nine patients underwent neurosurgical procedures, and six patients underwent EVD placement. Our case report and literature review have effectively demonstrated that outside the neonatal period, *Acinetobacter baumannii* meningitis was associated with neurosurgery and the placement of foreign material, such as an EVD.

The clinical manifestations of *Acinetobacter* meningitis are similar to those reported for meningitis. Most patients present with fever, convulsions, meningeal irritation, and

Table 3 Summary of *Acinetobacter* meningitis pediatric cases in the literature

Reference (year)	Age/sex	Underlying condition	Invasive procedure	Outcome
1. (1964) [11]	3.5 months/M	Hydrocephalus	Ventriculography	Survived
2. (1979) [12]	3 weeks/F	Myelomeningocele	Ventriculoperitoneal shunt	Survived
3. (1979) [13]	3 years/F	Medulloblastoma	Craniotomy	Survived
4. (1989) [14]	8 children	Leukemia	Intrathecal injections	3 died and 5 survived
5. (1990) [15]	4 years/UR	CSF leakage after head trauma	EVD	Survived
6. (1993) [16]	1 year/F	Astrocytoma	Craniotomy Ventriculostomy	Survived
	3 months/M	Medulloblastoma and hydrocephalus	Craniotomy Ventriculostomy	Survived
	2 months/F	Trauma hydrocephalus	Ventriculostomy	Survived
7. (1999) [17]	16 years/M	Hemangioblastoma	Ventriculostomy	Survived
8. (2002) [18]	14 years/M	Subdural hematoma	Extremal CSF shunt	Survived
9. (2006) [19]	4 years/M	Medulloblastoma	Craniotomy	Survived
10. (2008) [20]	3 years/M	Medulloblastoma	Craniotomy	Survived
11. (2009) [21]	2 months/F	Subarachnoid hemorrhage	EVD	Survived
12. (2010) [22]	3 years/F	Choroid plexus papilloma	Craniotomy and ventriculostomy	Survived
	14 years/F	Medulloblastoma	Craniotomy	Survived
	14 months/M	Congenital hydrocephalus	Ventriculostomy	Survived
13. (2011) [23]	9 years/F	Posterior fossa tumor	Craniotomy/EVD	Survived
	5 months/M	UR	EVD	Died
	9 years/M	Left frontal tumor	Craniotomy/EVD	Survived
	12 years/M	Road traffic injury	Craniotomy/EVD	Died
	9 months/M	UR	EVD	Survived
	3 months/F	UR	EVD	Survived
	2 months/F	UR	EVD	Survived
14. (2011) [24]	Neonate/UR	Myelomeningocele	EVD	Survived
15. (2012) [25]	15 years/M	Hydrocephalus Hypertensive nephrosclerosis	EVD	Survived
16. (2015) [26]	5 years/M	Medulloblastoma	Craniotomy	Survived

EVD external ventricular drainage, *UR* unreported

local signs of nervous system disturbances. The CSF typically shows an increased white blood cell count, mainly neutrophils, and biochemical indicators of decreased glucose levels and significantly increased protein levels. However, it is still necessary to exclude pseudomeningitis caused by CSF specimen contamination [27], as indicated by a positive CSF culture in the absence of clinical and laboratory features of meningitis. Two of our nine patients did not undergo neurosurgery but were included because these two children were neonates with fever, convulsions and bulging of the anterior fontanelle, and their CSF samples showed increases in the white cell count and protein level.

The appearance of *Acinetobacter* MDR, XDR and even PDR strains makes treatment very difficult and seriously threatens patient survival. Among our nine patients, most isolates were resistant to multiple classes of antimicrobials: there were four MDR cases, two XDR cases and one PDR case. In 7 of 8 patients in the retrospective studies by Saleem et al. [23], MDR *Acinetobacter baumannii* was cultured in the CSF. These case reports reflect the increasing resistance of *Acinetobacter baumannii*, and there may be no effective medicine in the future. In addition, *Acinetobacter baumannii* meningitis is usually associated with a high mortality, and the mortality rate of our 9 children approached 50%. Although whether the high mortality rate can be completely attributed to *Acinetobacter* infection is unclear and the severity of the intracranial injury itself also affects the prognosis, this mortality rate sounds the alarm for all pediatricians.

The rational selection of antibacterial drugs is crucial for neurosurgical *Acinetobacter baumannii* meningitis. For the first-line treatment, the carbapenem meropenem is the most commonly recommended empirical treatment, but its resistance rate exceeds 40% with carbapenem-resistant strains [28]. For such resistant *Acinetobacter*, polymyxin has been successful. However, polymyxin has a poor solubility in the CNS. When administered intravenously, its CSF level can only reach approximately 25% that of the serum level [18]; thus, it can be administered intrathecally or intraventricularly, combined with intravenous medications for these resistant *Acinetobacter* strains [29]. There are no unified standards for the intrathecal or intraventricular administration of polymyxin, and the relevant literature reports a wide range of doses. The recommended total daily dose should be from 0.75 to 7.5 mg colistin base activity (25,000–250,000 U polymyxin E sodium methanesulfonate) [19]. In 2004, the American Society of Infectious Diseases recommended a dose of intraventricular colistin of 125,000 U (10 mg) [30]. Possible complications of intraventricular or intrathecal administration include aseptic chemical meningitis or ventriculitis, but this neurotoxicity is dose-dependent and reversible [31].

Among our nine patients, two patients received colistin (5 mg/kg), and one received polymyxin B (2 mg/kg); their CSF cultures turned negative. Three patients received an intrathecal injection of amikacin (10–15 mg/day) according to the drug susceptibility test results, and two CSF culture turned negative; however, two patients still died. Therefore, the effect is not significant. Of the 4 patients who died, two still had positive CSF cultures, and the days to CSF sterilization of the other 2 patients were very high, with an average of 18.5 days. Thus, CSF sterilization is very difficult to achieve. However, this retrospective study still found many shortcomings in terms of clinical experience. There were discrepancies between our selection of antibiotics and the international recommendations. These discrepancies may be due to the rarity of pediatric *Acinetobacter baumannii* meningitis cases and the difficulty of obtaining polymyxins. Additionally, the polymyxin susceptibility tests were unconventional because our AST-GN16 susceptibility card does not routinely include polymyxin, and the bacterial laboratory does not have an additional polymyxin test strip. Few reports have described intrathecal antibiotic treatments, and there is no consensus regarding the dose. The treatment of *Acinetobacter* meningitis should last for at least 3 weeks [19] and should be thoroughly evaluated by clinical tests and repeated CSF cultures.

Conclusion

Acinetobacter baumannii meningitis occurs mostly after neurosurgery and is usually associated with the placement of foreign material, such as an EVD, and a high mortality rate. Most of our patients had cultured MDR strains. Therefore, antibiotic selection is challenging due to the appearance of MDR and even PDR bacteria. Early diagnosis and treatment are critical. However, it is very difficult to achieve CSF sterilization. Polymyxins are safe and effective for the treatment of *Acinetobacter baumannii* meningitis, and intrathecal or intraventricular administration is expected to be an effective choice for meningitis, but this drug still needs to be subjected to further clinical studies.

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Compliance with ethical standards

Availability of data and materials Data sharing is applicable to this article, and we wish to share our data.

Conflict of interest The authors declare that they have no competing interests.

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